purating and required cleaning out, but in none of them have I made any excision of the joint. In one case I broke the neck of the femur, but I can say that every one of them has been and is doing well. As to the function, this is, in all cases, considerably interfered with. Ankylosis is, as far as I know, the rule, and I can never forget the remark of Lorenz to me that ankylosis itself is a great safeguard after recovery from tubercular conditions. My cases are all straight.

In spite of the best possible treatment with mechanical apparatus, there are cases which will not get well and which run a course toward complete destruction of the joint; such cases require bone operative treatment.

STATISTICS, ETIOLOGY AND PATHOLOGY.

By E. H. WILEY, M. D., Los Angeles.

The subject under discussion labors under a variety of synonyms which are uselessly redundant and confusing. Thus we have morbus coxarius, morbus coxæ, hip disease, tuberculous disease of the hip, chronic articular osteitis of the hip, medulloarthritis, coxitis, coxalgia, and morbo coxario. The term, Hip Joint Disease, while not scientifically descriptive, is so well known and so commonly used that its application to the condition will not be easily supplanted. As a matter of fact, all these terms are used to cover a variety of conditions of tuberculosis, some of which do not even affect the joint, or affect it only secondarily.

We should naturally expect that tuberculous disease would most frequently attack the large joints most subject to use and most exposed to trauma. On account of the more sluggish circulation in dependent parts we should expect that the lower extremity would be most frequently the seat of the disease. Statistics bear out these premises, although they do not agree as to the relative frequency with which various joints are attacked. Thus, Young, compiling 1,000 cases, finds the vertebræ affected in 11.6 per cent, the hip in 42.1 per cent, and the knee in 10.3 per cent, while Waldvogel, compiling statistics of Prof. Koenig's cases between the years 1876 and 1895, finds the hip involved 568 and the knee 720 times. The preponderance of tuberculous infection of the knee seems theoretically more probable, considering the large size of the joint, its superficial situation and exposure to trauma, and its burden of weight with relatively poor static properties.

Most authorities agree that the right side is affected slightly more frequently than the left, the difference amounting to 1 per cent in Koenig's series, and slightly more according to some others. The difference may be accounted for by the greater frequency with which the right side is used and its slightly greater liability to sustain injuries.

The consideration of the etiology involves as an exciting cause the bacillus tuberculosis and various predisposing factors which deserve consideration.

Age—Hip Joint Disease is essentially one of childhood, though no age is exempt. By far the greatest number of cases occur before the age of 15 years. In Koenig's series 78 per cent occurred before this age. Of 5,461 cases noted by Knight, 88.2 per cent were under 14, and of 1,344 cases of Wright, Bryant and Sayre, 1,000 were under 15. This susceptibility during early years is due to the activity of joint growth, the liability of the joint to trauma, and to the large number of children within this age limit who are exposed to bad hygienic surroundings.

Sex—There is a slightly greater number of males affected, probably because the rougher habits of boys expose them to injury which is so often the determining factor in the localization of the disease.

Heredity—There can be no doubt that a tuberculous ancestry very strongly predisposes to hip disease. Of 229 cases of Koenig's in which a history was obtainable 35.4 per cent gave a tuberculous genesis. The real percentage is probably higher than is usually obtained, on account either of ignorance or of a disinclination to admit a family taint.

Traumatism—The principal factors in determining hip infection are those which lower the local power of resistance. Falls upon the trochanter or upon the feet, twists and wrenching injuries which impair the integrity of the joint very often precede the development of hip disease. It is interesting to note that it more often follows slight injuries than severe ones which fracture the bone or dislocate the joint. Statistics also note the fact that the process is not infrequently aggravated by traumatism.

Exanthematous diseases, which lower bodily resistance and which sometimes show a disposition toward localization in joints, are frequently followed by tuberculosis of the hip.

Pathogenesis—The hip joint well protected by soft tissues and not liable to open wounds, can only be attacked by tubercular infection in one of two ways: First, and most frequent, by way of the circulation, the local conditions being favorable for the development of the bacilli in or around the joint. Second, the extension of the process to the joint from infections of neighboring structures. It is also true that hip disease is usually secondary to a tuberculous focus elsewhere in the body. The commonest avenues of entrance are through the respiratory and intestinal tracts. In the first instance, enlarged and caseating cervical or bronchial glands usually result; in the second, strumous adenitis of the mesenteric or retroperitioneal groups. Other avenues of entrance, as the ear, are more rarely observed. Not infrequently the tuberculous primary area remains quiescent, or is only demonstrated post mortem.

Bearing in mind the fact that the blood is the carrier of the bacilli to the joint and its neighborhood, we can readily see that the localization of the process is determined by the vascular twig through which the bacillary embolus is propelled. Its entrance into the ramifications of the nutrient artery of the femur will be followed by the wedge-shaped bone infract with which we are familiar. On the

other hand, should the nutrient vessels of the asetabulum be the bearers, we should expect a primary acetabular process. In the same way the synovial membrane is often the seat of primary trouble. The bloodless cartilage should be exempt, and as a matter of fact, is spared a surprisingly long time after the mischief is well under way elsewhere in the joint. That other points should suffer with the hip by the same blood-borne infection is not strange, and Koenig's cases present thirty-five instances of bone and joint tuberculosis associated with the same process in the hip.

Pathology—While the late stages of the disease present an appearance familiar to all, the picture in the earlier stages varies materially with the location of the primary focus. The frequency with which the synovial membrane is primarily attacked is placed by Von Volkmann and Riedel as 16 to 17 per cent. One reason why it is difficult to estimate the occurrence of this form is the fact that statistics are gleaned from conditions present at operation, and those cases subjected to surgical interference have often suffered such extensive destructive changes that the positive identification of the primary focus is difficult or impossible.

The synovial form presents two types pathologically. The first, a mild form found almost altogether in children, characterized by a cloudy effusion into the joint, which contains more or less fibrin in masses. The synovialis itself is swollen, especially in the folds where it is reflected from the neck of the femur. It is moderately hyperæmic and presents areas of firm flat reddish granulation tissue which may also involve the head of the femur, without, however, doing serious damage to the cartilage upon which it lies. Occasionally a rarefying subchondral osteitis results in loosening the cartilage. The only indication of the tuberculous nature of the process is the presence upon or within the granulation masses of tubercules. This form, which, unfortunately, is not the most frequent, offers hope of retrogressive changes, leaving a functionally unimpaired joint. The far commoner form of synovial infection presents a sac filled by profuse and caseating granulations. The fatty acetabular tissues are swollen and the ligaments relaxed. The shallowed cavity allows of early luxations. Here follow secondary bone involvement and extensive destruction with the same appearance finally as in the primary bone necrosis.

Primary Bone Tuberculosis—When the focus appears first in the bone its location may be anywhere within, or in the neighborhood of the joint, and upon its location the further fate of the joint depends. The extra articular location carries with it the possibility of resolution without joint involvement. These places are: 1st, the trochanter major; this location is often seen in adults, while, 2d, in children the lower part of the trochanter minor is often selected; 3d, in the lower portion of the tuber ischii; 4th, in the ilium near the upper edge of the acetabulum; 5th, in the anterior inferior spine of the ilium. From these points the joint is secondarily infected by vascular or lymphatic channels with extensive synovial involvement, or remains free until an extension of the necrosis results in rupture into the cavity with widespread and rapid destruction.

The osseous lesion, primarily intra articular, may be situated either in the femur or in the acetabulum. These sites are infected with about equal frequency. They are more usually single, though often occurring in numbers. The favorite spots in the femur are beneath the articular cartilage or in the neck, while in acetabular foci, we find oftenest affected the upper and posterior edge of the cavity and the central triangle representing the junction of the three portions of the os innominatum. Osseous tuberculosis of the femoral head is not observed in young children, as the head is little ossified up to the fourth year of life. We then find in these children early involvement of the neck, which, on account of the extensive attachment of the capsules, is yet intra articular. The process is occasionally slow, with firm cartilageous granulations which cicatrize and shrink. This indolent form is known as tuberculosis sicca and tends to a spontaneous healing. The more ordinary process is distention of the joint with fluid and proliferation of granulation tissue which is primarily, or becomes secondarily tuberculous in character. The breaking down of this tissue, in which are larger or smaller sequestræ of bone forms the tuberculous abscess. The continuation of this destruction leaves bone ulcers. The cartilage meanwhile, after resisting for a considerable time, suffers by encroachment of granulation tissue from its surface, or from bone inflammation beneath, and may be loosened and when subjected to additional injury through movements of the joint, may ultimately come to lie loose within the cavity or even to be fragmented and absorbed entirely.

The increasing pressure within the joint, combined with the infiltration and destructive changes in its tissues, soon results in a rupture of the capsule and an extension of the process to the soft tissues. There is now present a cold, or tuberculous, abscess which finds its way to the surface and, rupturing, leaves a sinus or sinuses which tend to great chronicity.

According to the location of the focus and the rupture of the capsule, the subsequent appearance on the surface of the sinus may be anticipated. Thus, in anterior abscesses the pus emerges from the anterior and internal aspect of the capsule, after which it may pass to the outer side of the psoas magnus, or between the adductors and the vostus internus, or upward along the psoas tendon to the pelvis.

External abscesses originate after penetration of the bursa over the trochanter or from primary lesions of the trochanter itself.

The posterior group, following foci in the femoral neck or acetabulum pass downward to escape on the thigh above the lesser trochanter, or upward to spread over the ilium where they later point under the gluteal muscles. Sometimes these collections

burrow into the rectum and are thus discharged. Those bone abscesses originating in the acetabulum or ilium may also penetrate the pelvis and discharge themselves within it. There is always together with the destructive changes active tissue proliferation whose object is the limitation of the process here, as elsewhere, after discharge of tuberculous debris, by cicatrization. The triumph of this delimiting cicatrization, with the cessation of tuberculous destruction marks a fortunate ending to the process Its future, with the continuance of suppuration and tissue destruction, is followed by the deformity, sinus formation, emaciation and amyloid degeneration of various organs, whose termination is only brought about by the death of the unfortunate victim.

THE PROGNOSIS OF DIABETES.*

By EDWARD W. TWITCHELL, M. D., Sacramento.

Undoubtedly much of the ominousness with which the word diabetes is fraught, for the physician as well as the layman, is due to the time-honored attitude of the insurance societies and lodges towards those afflicted with that disease.

Whenever an individual applying for life insurance is discovered to have sugar in the urine he is rejected without more ado, and is furthermore disqualified for admission to other societies in the future. Consequently the public, lay and medical, has the habit of looking upon the diagnosis diabetes as equivalent to a death sentence shortly to be carried out.

There is no need to point out to you the fact that this attitude is anything but scientific, and not at all creditable to the profession. The layman is excusable, but the physician should no more take his ideas of prognosis from the insurance society than he should take his notions of therapeutics from the literature sent out by the drug houses. The factor of safety allowed by the insurance company is enough to warrant an expectation of a good many years for the average patient.

The pathology of the disease is not as yet on a sufficiently firm basis to allow one to differentiate the various clinical types from an anatomical standpoint, and we are constrained to group them according to their amenability to treatment. Using this as a basis for diagnosis looks like reasoning in a circle, but in the light of our present knowledge we can do no better.

Other things being equal, the younger the patient the severer the disease and the more quickly fatal. Most unfavorable are the cases in young adults of the poorer classes, who are poorly nourished at best, and who speedily go to pieces when the disease attacks them. An apparent exception to the rule must be noted in the fact that a certain number of infants and young children recover after a glycosuria running a very acute course. Aside from these few cases, the course of diabetes in the very young is extraordinarily rapid and fatal.

A number of acute cases ending in recovery are due to head injuries and as such are hardly to be classed in the same category with the pure diabetes.

The most favorable instances are found in the well-to-do and well-nourished adults past middle age, who can be induced to place themselves under proper dietary regime. In such cases the disease may run on for many years without any great inconvenience to the patient. In general, the disease may be looked upon as a chronic one, and if sugar elimination can be easily controlled by withdrawal of a moderate amount of carbo-hydrates from the diet, the progress of the ailment may be foretold as slow and gradual.

As to the possibility of recovery, this is exceedingly rare. Naunyn declares that he has no record of a single case of recovery where the disease has existed for any length of time, long enough, that is, to warrant the trouble being called chronic. Consequently the question of prognosis resolves itself into a guess as to the duration of the disease.

Very acute cases have been known to terminate fatally within five weeks. Wallach's case is interesting in this regard. The patient in question was a chemist who was in the habit of making weekly examinations of his urine. Five weeks before his death the urine was free from sugar. I am convinced from a recent experience of my own that many of these so-called acute cases are no more than chronic ones, long undiscovered, which have suddenly become aggravated. The patient of whom I speak was in coma and at the point of death when I first saw her. So little had her condition concerned her that the family physician was summoned only the day before. Careful inquiry after death made it pretty certain that she had been suffering from diabetes for a long period.

Statistics are, of course, no more reliable here than elsewhere, but it may be interesting to give some of those compiled by Naunyn. In 141 of his own patients, private and polyclinic, 42 died 1 year after recognition, 35 died between 1 and 2 years after recognition, 23 died between 2 and 3 years after recognition, 14 died between 3 and 4 years after recognition, 5 died between 4 and 5 years after recognition, 7 died between 5 and 6 years after recognition, 6 died between 6 and 8 years after recognition, 1 died between 8 and 10 years after recognition, 6 died between 10 and 12 years after recognition, 1 died 16 years after recognition, 1 died more than 31 years after recognition.

Of 64 severe cases, 59 died before the third year after discovery, and but one lived until the eighth year.

Naunyn remarks, however, that these statistics are hardly fair, as the disease in many instances may have existed for a long time prior to discovery. It may, none the less, be looked upon as a pretty accurate table of expectation.

From it we may deduce that while the severer cases usually end by the third year, the milder ones may drag on for thirty or even more.

Complications of all sorts may modify the picture to an extreme degree. A particularly fatal

* Read at the Thirty-seventh Annual Meeting of the State Society, Del Monte, April, 1907.